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Receptor-like Protein Tyrosine Phosphatase β (RPTP β) is Functious as a Receptor for *Helicobacter pylori* VacA

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Pathogenic strains of *Helicobacter pylori* produce a potent exotoxin, VacA, which causes progressive vacuolation and death of epithelial cells. We reported previously that VacA can interact with target cells by binding to a 250-kDa receptor protein, receptor-like protein tyrosine phosphatase β (RPTP β) (1,2) as well as that agents that promote differentiation of HL-60 cells into macrophage-like and monocyte-like, but not granulocyte-like, cells, enhanced VacA sensitivity by increasing expression of cell surface RPTP β (3).

To define further whether RPTP β is functious as the VacA receptor and mediates vacuolation, human RPTP β cDNA were prepared in a pBK-CMV-vector. The RPTP β genes were transfected into BHK-21 cells, which are insensitive to VacA. Expression of cell surface RPTP β and VacA-sensitivity of the transfected cells were quantified by FACScan analysis and Neutral Red Uptake assays. The presence on the cell surface of RPTP β protein resulted in the induction of VacA sensitivity.

These data support the hypothesis that RPTP β is functious as a receptor for VacA.

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